Lesson of the Week

Re-expansion pulmonary oedema: a potentially serious complication of delayed diagnosis of pneumothorax

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Re-expansion pulmonary oedema after intercostal drainage of pneumothorax is a well documented condition with serious consequences that are not widely appreciated. Delayed diagnosis and treatment predispose to the condition, and its consequences may be exacerbated by inappropriate management. We describe two cases.

Re-expansion pulmonary oedema may develop if diagnosis and treatment of pneumothorax are delayed. This condition may be fatal if inappropriately managed

Case reports

CASE 1

A 14 year old boy with a history of mild atopic asthma was admitted with left sided pleuritic chest pain and dyspnoea. These symptoms had first been noticed five days before admission. He had been seen by his general practitioner and prescribed antibiotics. His symptoms had persisted and then worsened, at which point he was admitted. Clinical examination and chest radiography showed a large left sided tension pneumothorax. A drain was inserted through the second left interspace anteriorly and connected to an underwater seal. No suction was applied. There was a rapid egress of air, and radiography confirmed almost complete re-expansion of the lung while also showing interstitial shadowing on the left (fig 1).

Examination disclosed widespread crackles throughout the left lung with occasional wheezes. Blood pressure fell from 120/70 mm Hg on admission to 100/60 mm Hg when measured two and six hours later, and his heart rate rose from 96 to 115 beats/minute. Records of fluid balance showed an intake of 1840 ml and urine output of 400 ml during the first 16 hours. During the next day a diuresis was observed with an intake of 1650 ml and urine output of 1900 ml. His pulse and blood pressure returned to normal, and chest radiographic findings gradually improved, with almost complete resolution of the pulmonary oedema after seven days. Two and four months later he suffered two further left sided pneumothoraces. On each occasion he was treated with intercostal tube drainage within 24 hours and unilateral pulmonary oedema was not observed.

CASE 2

Four weeks before admission a 40 year old woman presented to her general practitioner with right sided pleuritic chest pain, non-productive cough, and breathlessness. She smoked 15 cigarettes a day but did not have a history of chest disease. She was given an antibiotic and an expectorant. Her symptoms worsened, and she was admitted. Examination and chest radiography showed a large right sided tension pneumothorax. An intercostal drain was inserted through the seventh right interspace in the midaxillary line. Some relief of dyspnoea was achieved as some air escaped through an underwater seal drain. Chest radiography showed only minimal re-expansion of the right lung and 12 hours later showed a tension pneumothorax.

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In an attempt to relieve blockage the intercostal tube was withdrawn a few centimetres, which resulted in vigorous bubbling of air through the underwater seal. No suction was applied. Immediately after this she became extremely distressed, breathless, sweaty, and cyanosed. She coughed up frothy, bloodstained sputum. Her hands and feet felt cold, her blood pressure fell from 120/70 mm Hg to 80/65 mm Hg, and she had a tachycardia of 110 beats/minute. Her arterial blood tensions on 40% oxygen were oxygen

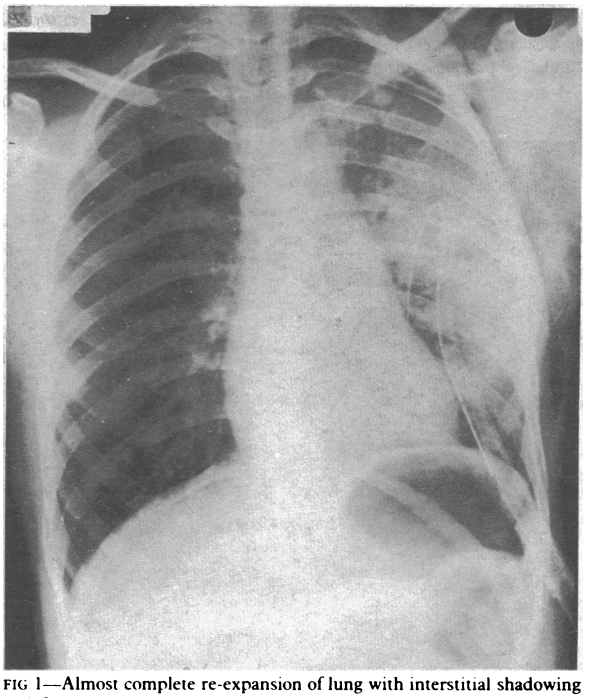

FIG 1—Almost complete re-expansion of lung with interstitial shadowing on left.

9·3 kPa (69 mm Hg) and carbon dioxide 5·3 kPa (40 mm Hg). A chest radiograph showed complete expansion of the right lung with changes consistent with unilateral pulmonary oedema (fig 2). An electrocardiogram was normal. Immediate resuscitation was started with intravenous plasma and physiological saline.

Within five hours the blood pressure had returned to normal and her heart rate settled to 90 beats/minute. She became well perfused peripherally, and urine output and plasma urea and electrolyte concentrations remained satisfactory. Despite the improvement in her haemodynamic state she remained hypoxic with an oxygen tension of 9·3 kPa (69 mm Hg) on 40% inspired oxygen. A radiograph taken the next day showed that a partial

pneumothorax had recurred on the right side and the right lung continued to show pulmonary oedema. In addition the left side showed pulmonary oedema (fig 3). A second drain was inserted on the right in an attempt to achieve full re-expansion. Suction limited to 10 cm water was applied, and complete re-expansion was eventually obtained.

Over the next four days the pulmonary oedema resolved without further treatment. She was discharged eight days after her initial admission but was readmitted two weeks later with another right sided pneumothorax. Complete re-expansion was achieved by applying negative pressure of 8 cm water. No pulmonary oedema resulted. In view of her past problems a talc pleurodesis was performed.

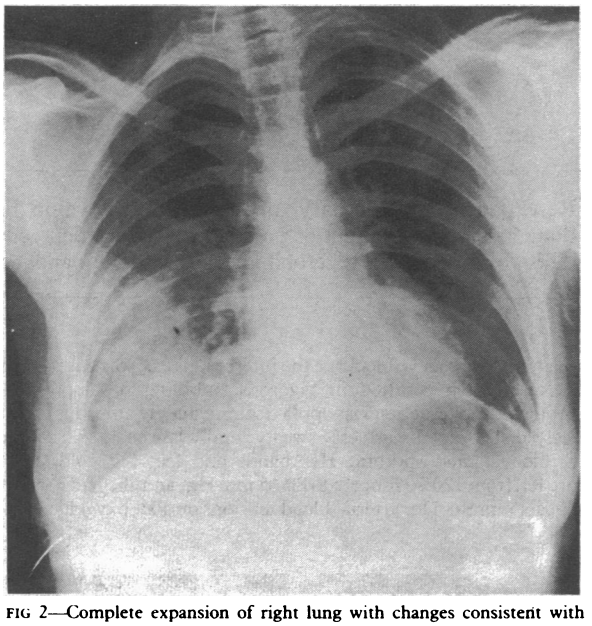

FIG 2—Complete expansion of right lung with changes consistent with unilateral pulmonary oedema.

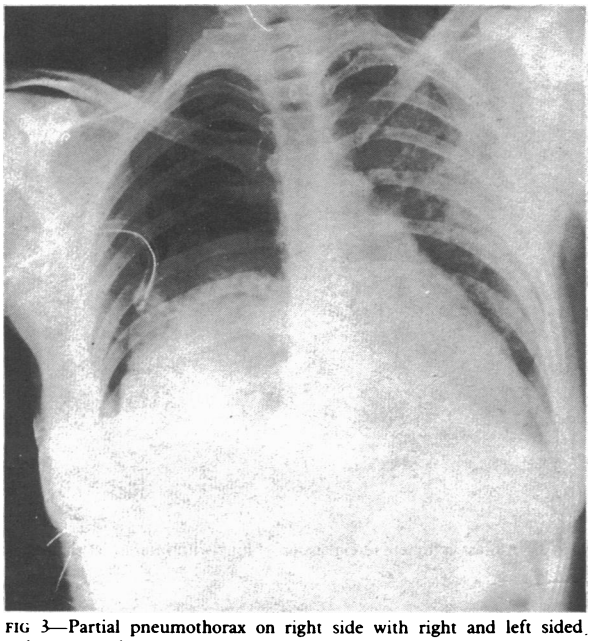

FIG 3—Partial pneumothorax on right side with right and left sided pulmonary oedema.

Discussion

Pulmonary oedema occurring in a re-expanded lung after aspiration of a pleural effusion was first described by Foucart in 1875 (cited by Carlson *et al*[1]). A series of 42 cases of "albuminous expectoration" was reported in 1905.[2] It is now widely recognised that this complication may occur if excessive volumes of pleural fluid are removed at one time and that the risk of this complication may be reduced by limiting the volume of fluid aspirated. It is less well known that this complication may complicate the management of spontaneous pneumothorax, as first described in 1959[1]; sporadic cases have been reported subsequently and were reviewed by Kassis *et al* in 1981.[3] Out of 19 recorded cases, 16 were in subjects whose lung had been totally collapsed for at least three days.

In our two cases the first incident of pneumothorax probably occurred five days and four weeks before treatment, respectively; when subsequent episodes of pneumothorax were treated promptly re-expansion pulmonary oedema did not develop.

The pathogenesis of re-expansion pulmonary oedema is a matter of some debate. Early reports placed emphasis on the role of excessive use of intrapleural suction,[4,5] but this was clearly not implicated in our cases or those of several other authors,[3,6-9] although excessive negative intrapleural pressure may have been responsible for the condition when it occurred despite early treatment.[3] The observation that re-expansion pulmonary oedema most commonly develops in lungs that have been totally collapsed for several days suggests that damage occurs to the capillary-alveolar barrier during this time. Anoxia may perhaps lead to increased pulmonary capillary permeability,[10,11] and loss of surfactant is a further possibility.[7]

An unusual feature in our second case was the development of contralateral oedema. This has not previously been reported in man, although it was seen in an animal model.[12] Its development in our patient was delayed for 24 hours, and it may have evolved through a different mechanism; possibly it represented the supervention of the adult respiratory distress syndrome. Whatever the mechanism of production of oedema, the effect is one of acute hypovolaemia as evidenced in both our cases by hypotension and tachycardia and in case 1 by oliguria. In case 2 urine output was maintained after infusion of intravenous fluids. We believe that this condition is not widely known and might be treated by inexperienced physicians with diuretics, which would exacerbate the condition. In one case this was done with fatal results.[7]

We hope that as physicians become more aware of the condition patients will be referred for chest radiography early. Inevitably, however, some patients will present with a history suggesting that the pneumothorax developed some days earlier. How should these be managed? We doubt whether it is feasible to insert an intercostal tube and clamp it as would be the case in controlling drainage of a pleural effusion. The egress of air is difficult to control, and clamping the drain tube would favour the development of surgical emphysema. We suggest that in patients who present late with pneumothoraces simple aspiration using the method described by Hamilton and Archer[13] should be considered, as this procedure can be stopped if the patient shows any sign of distress.

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